

which the bacilli are found in the circulation is really greater than in other conditions (puerperal sepsis, ulcerative endocarditis, etc.) which are generally so recognized.

While the usual portal of infection is probably the gastro-intestinal tract, the local lesions produced at the point of entry may, in some instances, be slight. In other cases it is possible that, as in some streptococcus infections, the processes may remain local, being arrested by the lymphatic apparatus of the intestine and the retroperitoneal glands. In most cases, however, the parasites pass "the protecting wall of the mesenteric glands," causing a general infection of the lymphatic system and the blood just in the same manner as a general infection occurs after wound infection. By the blood the bacilli are distributed throughout the organism, producing more or less characteristic manifestations. The author is inclined to even believe that the tendency toward involvement of the intestinal lymphatic apparatus is not necessarily the result of the fact that the intestine is the ordinary portal of entry, citing in support of this view the involvement of this system in relapses where it is much more reasonable to believe that the renewed infection springs from depots within the organism and from the intestinal tract as well as the fact that many areas far apart are so often simultaneously affected. He is inclined rather to assume that the intestinal follicles have a special predisposition toward infection with the typhoid organisms even when present in the general circulation.

[These latter theories have much in their support. If the general medical public only realized the fact that typhoid fever is clinically a general septicæmia, of which the intestinal changes form the more marked local manifestations, and not an intestinal disease, the senselessness of attempts at aborting or influencing this malady by so-called intestinal antiseptics would be more readily appreciated.—W. S. T.]

A Case of Typhoid Fever without Intestinal Lesions.—BLUMENTHAL (*Deutsche med. Wochenschr.*, 1902, xxviii. 626) from Stadlemann's clinic, in Berlin, reports the case of a woman, aged twenty-three years, who entered the hospital on the tenth day of typhoid fever. She was at that time five months' pregnant. The symptoms were characteristic; well-marked roseola and Diazo reaction were present, while the Widal reaction was positive at a dilution of 1/150. On the fourteenth day the patient aborted, and on the twentieth, died. At autopsy there was a hemorrhagic enteritis of moderate degree in the jejunum. In the lower part of the ileum the follicles were a little prominent; no affection of Peyer's patches. The ileocecal glands were enlarged, on section, soft and grayish red, with prominent follicles; the spleen was large and soft. Cultures from the spleen and glands showed numerous bacilli answering to all the characteristics of the typhoid organism.

[This case emphasizes again the fact that the essential symptoms of typhoid fever are not dependent upon the intestinal lesions, but rather those of a general septicæmia with certain special characteristics.—W. S. T.]

Further Observations on the Relation of Changes in the Pancreas to Diabetes.—WEICHSELBAUM and STENGEL (*Wiener klin. Wochenschr.*, 1902, xv. 969) contribute an interesting discussion to this important question.

After considering the important contributions of Ople, Herzog, Wright and Joslin, Schmidt, von Hanseemann, Sobolew and Schultz, they discuss seventeen observations which they are able to add to their eighteen previously reported. Two of these cases were instances in which, clinically, the condition was regarded not as pancreatic diabetes, but as glycosuria of other origin. In the other fifteen cases the islands of Langerhans were found on necropsy to be diminished in number in every instance; sometimes so markedly that in large sections none could be found, while those islands which remained showed not only diminution in size, but "constant, definite, unmistakable changes." These were of three types: (a) Simple atrophy; (b) vacuolization or liquefaction; (c) an increase in connective tissue. In three cases hemorrhage into several of the islands was found, while twice marked deposition of fat granules, especially limited to the islands, was made out. These changes in the islands cannot, in all instances, be secondary to sclerotic changes elsewhere in the pancreas.

The proper interpretation of these observations and those of the authors previously noted is discussed at some length. v. Hanseemann, an opponent of the theory that disease of the islands of Langerhans stands in direct causal relation to diabetes, noted that in 34 cases they were never wholly absent, while in only 6 was a sclerosis to be made out. But this argument is hardly sufficient in view of the mass of observations which have been accumulated; for the absence or disease of the majority of islands might well seriously injure their function, while, if one were to suppose that a total destruction alone resulted in diabetes, it would be difficult to explain the frequent improvements or recoveries which are seen. v. Hanseemann himself observes that he has never seen a sclerosis of the islands without diabetes. It must be remembered, too, that sclerosis is by no means the only or even the commonest change which is found in the pancreas of diabetics. The authors conclude that there is much in favor of the theory that on the one hand Langerhans' islands play a most important rôle in the metabolism of the carbohydrates, while, on the other hand, pancreatic diabetes may result from a disease of these islands. Their conclusions are as follows:

1. In all cases of pancreatic diabetes, excepting those which they have clinically ruled out as not belonging to this class, they have found constant definite changes in the islands, changes sufficient to impair or destroy their function. These changes are not present in non-diabetics. In the non-diabetic atrophy of the pancreas in general marasmus they were able to make out a marked thinning of the epithelium, but neither liquefaction, destruction, nor diminution in the number of the islands.

2. The authors quoted above have found in all or in a considerable majority of their cases similar or like changes.

3. In these cases of pancreatic diabetes the remaining pancreatic tissue shows no changes or, at all events, alterations which are appreciably less marked than those of the islands, and are of a nature which from the experimental investigations of von Mehling and Minkowsky would have been insufficient to cause diabetes.

4. Those cases of glycosuria in which no changes in the islands have been found, admit always of an explanation which is reconcilable with the above-mentioned theory.

5. The histogenesis and structure of the islands make it probable that they have a wholly different function from that of the true gland parenchyma of the pancreas.

6. The experimental observations of Sobolew, Schultze, as well as one of their own cases, show that the islands behave in an entirely different manner toward the agent producing pancreatic diabetes than toward those poisons which produce the other forms of pancreatic atrophy.

[These observations wholly confirm those of Opie demonstrating the remarkable—very probably causal—relation between disease of the islands of Langerhans and diabetes. It must, however, be remembered that, as Opie has pointed out, there are instances of diabetes in which no changes in the pancreas are demonstrable—and it must be acknowledged that we have as yet no methods for recognizing these two classes of cases clinically.—W. S. T.]

Eosinophilia in Filariasis.—CALVERT (*Johns Hopkins Hospital Bulletin*, June, 1902, p. 133), in studying the blood in four cases of filariasis in natives of the Island of Luzon, P. I., found that there was a quite marked eosinophilia in all cases. His attention was drawn to the possibility of an eosinophilia occurring in filariasis by the finding of enormous numbers of eosinophiles in the tissues surrounding the lymph vessels and in the bloodvessels of a patient who died of plague, but who was found at autopsy to have filariasis. Subsequently three cases were studied clinically, and an increase of the eosinophiles found in all. The highest eosinophile count was 22 per cent. In all the cases there was also a leucocytosis. An interesting point brought out was that the eosinophilia was most marked at the time of the day when the peripheral circulation was free from embryo filariæ. The leucocytosis and eosinophilia are apparently present only in the early stages of the disease, both disappearing in chronic cases.

The Etiology of the Summer Diarrhœa of Infants; A Preliminary Report.—A short time ago it was noted in this department that Vedder and Duval had investigated isolated cases of dysentery in the Philadelphia hospitals, as well as epidemics occurring in institutions in Lancaster County, Pennsylvania, and at New Haven, Connecticut. They had found, as a result of their study, that the dysentery was produced by *bacillus dysentericæ*, the same organism that had been isolated from cases of acute dysentery in adults by Shiga in Japan, Flexner and Strong in the Philippines, Kruse in Germany. They seemed justified in concluding that this organism was the cause of acute dysentery the world over.

DUVAL and BASSETT (*American Medicine*, September 13, 1902, p. 417) have just made a preliminary report on their bacteriological studies of the cases of summer diarrhœas of infants admitted to the Thomas Wilson Sanitarium, near Baltimore, Maryland. They isolated *bacillus dysentericæ* Shiga from the stools of forty-two of these cases. The organism was also found in the scrapings of the intestinal mucosa, and in one case from the mesenteric glands and liver. From an etiological and therapeutical standpoint this important discovery places the summer diarrhœa of infants on the same basis with the acute dysenteries of adults.